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THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 33 Number 11

November 15, 1949

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station — Beltsville, Maryland
Volume 33 — November 15, 1949 — Number 11

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THE PLANT DISEASE REPORTER TO BE MULTILITHED

Beginning with January 1950 the Reporter will be multilithed. We know that this will please all of our contributors and readers. The new form of publication will add greatly to the usefulness of the Reporter, besides making it much more attractive in appearance.

This is an opportune time to explain the limitations of our method of publication, and to make some suggestions about the preparation of manuscripts for the Reporter.

Tables and Illustrations

When planning tables or illustrations for use in a Reporter article, contributors should keep in mind, first, the size of the page, which for text, tables or illustrations, including footnotes, titles, and legends, is not more than 6 inches by 8 3/4 inches; second, the typographical limitations that do not permit small type for tables.

With the multilith process tables and illustrations can be reduced, but not so much as to decrease legibility. The extra step of reduction adds to the expense of the Reporter, and whenever possible, both tables and illustrations should be designed to fit the Reporter page without change of size. Probably the content of most large complicated tables would gain in interest and clarity if divided into smaller units requiring less study to understand.

Photographs must be clearly focused, with good contrast, to reproduce well by the multilith process.

Maps, charts, and graphs must be clearly drawn with black ink, with clear symbols and lettering of a size to be legible if reduction is necessary. Remember that on maps and graph paper, blue boundary lines, blue base lines, etc., will photograph out. Orange or green will stay in without special filters, and you should let us know whether or not you wish the background lines to be retained. On blue-lined paper, any base lines that you wish to show should be inked in. Clear glossy photographs of the original drawings can be used.

If typing is used for legends, etc., it should be black and clear, without erasures. Typing should not be used if much reduction is necessary.

We must have finished drawings, or glossy prints, for multilithing. It will no longer be possible to reproduce from pencil drawings or from mimeographed copy.

Schedule of Publication

You might like to know our schedule for getting out the Reporter. The deadline for receiving articles is the eighteenth of the month preceding the date of issue, i. e., for an article to appear in this November 15 number, it must have reached us by October 18. The only exception to this rule is for short announcements or brief emergency reports received before the end of the month.

Thus: copy is closed on the eighteenth, the completed typescript is sent for duplicating at the end of the month, and the Reporter comes out on the fifteenth of the following month.

Trademarks

More than once, recently, the company concerned has objected to the incorrect use of a trademarked name in a Reporter article. Of course this is important. Use of a proprietary name as the equivalent of a common name for similar products, or ascribing the name to the wrong manufacturer, is injurious to the manufacturer's interests. Please be sure to use such names in their exclusive sense, and if you mention the manufacturer or chemical composition be sure you give them correctly.

Conclusion

Of course it is content that is really important. The Reporter fills a special need in plant disease literature. Anything of timely active interest in plant pathology is its field, ranging all the way from history to prophecy. History catches up with prophecy rapidly in this changing world, and the Reporter's specific advantage of prompt publication gives a chance to keep abreast or even a little ahead of history.

Preliminary results, tentative conclusions, or speculation can be set forth while really new and therefore most useful, even though not complete or definite enough for more formal presentation. Reports of unusual outbreaks or of newly discovered diseases help to forestall threatening development. Special surveys add to knowledge of plant disease geography and ecology. Historical summaries correlate older discoveries with new knowledge and often suggest the direction of future study.

Geography, environmental relations, spread, newly discovered diseases, new developments in control, damage and technique of appraisal--all of these are topics for discussion in the Reporter. We are proud to be associated with our contributors in maintaining this service.

DIVISION OF MYCOLOGY AND DISEASE SURVEY

NOTICE OF HEARING ON "TOLERANCES FOR POISONOUS OR DELETERIOUS RESIDUES ON OR IN FRESH FRUITS AND VEGETABLES"

A public hearing upon proposals to issue regulations limiting the quantity of poisonous or deleterious residue on or in fresh fruits and fresh vegetables and to amend or to repeal the regulation limiting the quantity of fluorine remaining as insecticidal residue on apples and pears has been announced by the Federal Security Agency, Food and Drug Administration (Federal Register, Sept. 17, 1949: 14 F. R. 5724). The hearing will commence at 10:00 a.m., January 17, 1950, in room 5140, Federal Security Building, Third Street and Independence Avenue, S. W., Washington, D. C.

The notice states that the proposals, in general terms, are as follows:

"First, to take evidence on the following points:

"1. Which fruits and vegetables require the addition or application of a poisonous or deleterious substance in their commercial production.

"2. What poisonous or deleterious substances are required on each such fruit and vegetable for its commercial production.

"3. To what extent can such substances be avoided by washing, cleaning, or otherwise removing residues from the fruits and vegetables before marketing.

"4. The quantity of each poisonous or deleterious substance that can be tolerated on each such fresh fruit or fresh vegetable or on classes of fresh fruits and fresh vegetables without danger to public health, taking into consideration the other ways in which the consumer may be affected by the same or other poisonous or deleterious substances from other sources.

"Second, on the basis of such evidence (if it so indicates), it is proposed to promulgate regulations limiting the quantity of such poisonous or deleterious substances as are required on or in such fruits and vegetables, individually or by classes, the amount of the various substances tolerated to be fixed in terms of parts by weight, or by setting such other limits as is shown by the evidence to be necessary for the protection of public health.

"Third, to repeal or to so amend the regulation limiting the quantity of fluorine remaining as insecticidal residue on apples and pears (21 CFR, 1944 Supp., 120.1), as to bring it into harmony with other regulations adopted.

"For the purposes of the hearing on the points outlined above, evidence will be taken on any insecticidal or fungicidal substance, exclusive of inert ingredients therein, used on any fruit or vegetable."

Plant pathologists will see the opportunity for a review of the whole subject from the standpoint of possible fungicidal toxicity in the light of present-day recommendations for use of some of the newer fungicides, and the desirability of having all available evidence presented at the hearing.

Tom Bellis of the Food and Drug Administration, Division of State Cooperation, has outlined the procedure prescribed for the Administration in establishing regulations ("Rule-making under 406(2) of the Federal Food, Drug, and Cosmetic Act," talk presented at the meeting of the National Agricultural Chemicals Association, Spring Lake, New Jersey, September 9, 1949). Briefly, the procedure includes announcement of a hearing on the initiative of the Administrator or upon an application stating reasonable grounds from an interested industry, to be published not less than 30 days before the date of the hearing. At the hearing evidence is taken from witnesses testifying under oath and subject to cross-examination. Witnesses should have at least five copies of each exhibit which they intend to offer in evidence, and it is good practice to have several additional copies for distribution at the meeting.

Further: "All testimony is recorded by a private reporting firm. The firm which has the contract for this fiscal year is the Acme Reporting Company of Washington, D. C. and they sell copies of the transcript. Parties who wish to buy copies make their arrangements with the reporter at the hearing. The presiding officer routinely affords the parties of record an opportunity to present for the Administrator's consideration recommended findings of fact, and suggested regulations. These are written and are accompanied by briefs.

"After the record has been studied and the suggested findings and regulations considered, the Administrator publishes in the Federal Register a tentative order incorporating the rules which he proposes to make. The tentative order appears in that part of the Federal Register headed 'Proposed Rule-Making'. To this tentative order parties of record who may have exceptions are given an opportunity to file them and briefs in support of their exceptions. These exceptions are considered and then a final order is published. The final order carries a notification of the date when the regulations established by the order will take effect. Unless an emergency situation exists, the effective date is not less than 90 days following the issuance of the final order. Normally this ends the rule-making procedure."

To sum up, rule-making is not an arbitrary act, but arises from necessity for regulation; and is based on all the evidence that can be acquired, while the tentative order gives opportunity for trying out and revision before the final order is promulgated.

The Food and Drug Administration needs all the information it can get, on all the aspects of this toxicity question. Plant pathologists have a responsibility to contribute their experience.

THE USE OF SODIUM HYPOCHLORITE FOR THE CONTROL OF THE BROWN ROT DISEASE OF PEACH IN DELAWARE[1]

Panos L. Poulos[2]

New compounds are constantly being sought by this and other experiment stations for use as fungicides for the control of the brown rot disease of peach. A commercial solution of sodium hypochlorite (Clorox, 5.25% active ingredient) was included among the compounds tested during the spring and summer of 1949.

The use of sodium hypochlorite as a fungicide is not new, but it is new as far as use in controlling brown rot of peach. Baker and Heald (1) in 1932 tested Westchlor on the viability of blue mold spores on apples. They used it as a spray and dust in Washington and recommended its use for control of blue mold on apple. Weindling (4) in 1940 obtained favorable results using sodium hypochlorite as a spray and dust for treating cotton seed for protection against damping-off caused by Glomerella gossypii. Cardinell and Mitchell (2) in Michigan in 1947 employed an "F.M.C. Steri-cooler" in which a chlorine-type germicide (Hypo-clor) was added to control brown rot in the package.

In a recent issue of the Plant Disease Reporter, Mason (3) reported favorable results when a commercial solution of sodium hypochlorite (Clorox) was used as a spray in Arkansas for the control of the bacterial spot [Xanthomonas pruni] and the brown rot disease [Monilinia fructicola] of peach. As the data obtained from experimentation with sodium hypochlorite (Clorox) in Delaware are contradictory to those obtained in Arkansas, this article is submitted for consideration of plant pathologists working with fungicides on peaches.

Phytotoxicity tests were conducted on June 3 and 9 at the University Farm using Clorox to determine what concentrations would be safe for use as a pre-harvest spray. Concentrations ranging from 64.5 to 2000 ppm Cl were tested on the Summer Crest, White Hale, and Fisher varieties. Concentrations ranging from 250 to 2000 ppm Cl (1 to 8 gals. Clorox per 100 gals. water) produced bleaching symptoms on the foliage two to five days after the first application. The young fruit was not injured at any concentration. The average temperature at the time of application was 86°F. and the humidity was high.

Experiments were conducted during the pre-harvest period using sodium hypochlorite (Clorox) at a concentration of 100 ppm Cl (0.4 gal. Clorox to 100 gallons water) on the Early Redfree, Halehaven, and Elberta

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[1] Published as Miscellaneous Paper No. 73 with the approval of the Director of the Delaware Agricultural Experiment Station. Contribution No. 19 of the Department of Plant Pathology.

[2] Assistant Research Professor.

Table 1. Sodium hypochlorite (Clorox) as a pre-harvest spray for the control of brown rot in Delaware.

Treatment	Conc./100 gal.	ppm Cl	Percentage Brown Rot		
			Early Redfree[a]	Halehaven[b]	Elberta[c]
Untreated	---	---	36.0	8.0	20.0
Clorox	0.4 gal.	100	45.0	14.0	19.0
Clorox + Sulforon X	0.4 gal. - 3 lbs.	100	39.0	7.0	20.0
L.S.D. at 5%				7.8	13.5

[a] Two applications: June 21, 29. Scored: July 5.
[b] Three applications: July 14, 19, 23. Scored: July 24.
[c] Five applications: July 18, 24, 29, Aug. 4, 8. Scored: Aug. 8, 11.

Table 2. Sodium hypochlorite (Clorox) as a chemical dip for the control of the brown rot disease of peach in the package.

Treatment[a]	Conc./100 gal.	ppm Cl	Percentage Brown Rot		
			Triogem[b]	Halehaven[c]	Elberta[d]
Untreated	---	---	29.4	82.4	30.4
Water Dip	---	---	34.5	87.4	60.8
Liquid lime sulfur	2 qts.	---	15.1	48.5	17.7
Liquid lime sulfur + Clorox	2 qts. - 4 gal.	1000	---	50.7	---
Clorox	16 gal.	4000	---	56.9	56.5
ditto	8 gal.	2000	---	63.6	47.0
ditto	8 gal.	2000	---	54.4	36.5
ditto	4 gal.	1000	25.3	59.0	26.5
ditto	2 gal.	500	22.8	---	---
ditto	1 gal.	250	27.3	---	---
L.S.D. at 5% Level			12.8	18.7	8.0

[a] Peaches brushed, graded, dipped for 30 seconds, drained for 5 minutes, placed in open-staved half bushel baskets with liner and lid, stored at room temperature for four days.
[b] Dipped: July 19. Scored: July 22.
[c] Dipped: July 25. Scored: July 29.
[d] Dipped: August 15. Scored: August 19.
[e] Dipped for five minutes.

varieties. The Early Redfree variety was located at Rising Sun, and the Halehaven and Elberta varieties at Bridgeville, Delaware. The average temperatures throughout the pre-harvest period were 85° to 90°F. at the times of application. Rainfall was very light but the humidity was high. There was no indication of foliage injury. Complete data are presented in Table 1. These data indicate that sodium hypochlorite (Clorox) did not control brown rot under the conditions of the experiments in Delaware.

In view of the fact that sodium hypochlorite (Clorox) was not toxic to the fruit at any of the concentrations tested, it was included in chemical dip tests on three varieties for the control of brown rot in the package. Concentrations ranging from 250 to 4000 ppm Cl (1.0 to 16.0 gal. Clorox to 100 gal. water) were tested. These peaches were brushed, graded, dipped for one minute, drained for five minutes, packed in open-staved half-bushel baskets with liner and lid and stored at room temperature (approximately 80°F.) for four days. At the end of four days the peaches were scored for rot control. At concentrations of 250 to 1000 ppm Cl (1.0 to 4.0 gal. Clorox to 100 gal. water) the color of the peach was enhanced. Concentrations of 2000 to 4000 ppm Cl (8-16 gal. Clorox to 100 gal. water) caused a slight brown mottling on the surface of the peach. Complete data are presented in Table 2.

The data in Table 2 show that sodium hypochlorite (Clorox) is not effective as a chemical dip for the control of the brown rot disease of peach in the package.

Literature cited

1. Baker, K. F. and F. D. Heald. Some problems concerning blue mold in relation to cleaning and packing apples. Phytopath. 22 (11): 879-898. 1932.
2. Cardinell, H. A. and A. E. Mitchell. Packing house trials to reduce peach-rot. Michigan Agr. Exp. Sta. Quar. Bull. 30 (4): 460-467. 1948.
3. Mason, C. L. The use of sodium hypochlorite in the control of peach diseases. Pl. Dis. Reptr. 33 (8): 319-320. 1949.
4. Weindling, R. Phytopathological notes. Sodium hypochlorite shows promise as a seed treatment. Phytopath. 30 (12): 1051-1052. 1940.

DELAWARE AGRICULTURAL EXPERIMENT STATION, DEPARTMENT OF PLANT PATHOLOGY, NEWARK, DELAWARE

IDENTITY OF ANTHRACNOSE OF LUPINE AND PEACH CAUSED BY GLOMERELLA CINGULATA[1]

J. L. Weimer and J. C. Dunegan[2]

Anthracnose of peaches shipped from Georgia was reported by Ramsey, Smith, and Heiberg (2) in 1948. They found the causal fungus capable of attacking apple, pear, and plum, as well as peach fruits. More recently, Dunegan and Kephart (1) reported peach anthracnose on fruit from Georgia and Maryland. Lupine (Lupinus angustifolius L.) anthracnose was described by Weimer (3) in 1943 as being caused by Glomerella cingulata (Stonem.) Spauld. & Schrenk. This fungus could not be distinguished from isolates of G. cingulata from apples, either morphologically or in their ability to decay apple fruit. Since the blue lupine is now planted extensively as a winter cover crop in and near peach orchards in Georgia, and since both lupine and peach are attacked by G. cingulata, it seemed desirable to determine definitely whether strains of the fungus from one of these hosts can infect the other. With this in mind, cross inoculations on peach and lupine were made with isolates of the fungus from both hosts.

On February 18, 1949, spores from two isolates of the fungus, G. cingulata, one from peach and one from lupine, were atomized onto young blue lupine plants growing in sterilized soil in the greenhouse. Control plants were atomized with sterile tap water. After being inoculated, the plants were held in a moist chamber for 68 hours and were then returned to the greenhouse bench. Early stages of infection were observed on the petioles of inoculated plants within 68 hours. On February 25, all of the petioles of the plants inoculated with the fungus from peach were dead, and typical anthracnose lesions were present on the leaflets. Infection of the plants inoculated with the isolate from lupine was unmistakable, but the lesions were less abundant because the isolate produced relatively few spores. There were no anthracnose lesions on the control plants.

On March 28, 1949, further inoculations were made by using the same method. In addition to the two isolates, one from lupine and one from peach, used previously, a reisolate of the fungus from lesions on a lupine plant inoculated with the isolate from peach in the earlier experiment was used. The inoculated lupine plants and controls were held in the moist chamber for 48 hours. Observations made on April 1 showed that the plants inoculated with the isolate from peach were heavily infected. Stems, petioles, and leaflets on most of the inoculated

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[1] Paper No. 189, Journal Series, Georgia Agricultural Experiment Station.

[2] Cooperative investigation between the Division of Forage Crops and Diseases and the Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering; and the Georgia Agricultural Experiment Station.

plants showed many typical anthracnose lesions. By May 6, 21 out of 28 plants inoculated with the peach isolate were diseased, the remainder being healthy. Of the 21, 12 were dead and the others were more or less severely injured. One of the 10 plants inoculted with the reisolate from the previous experiment remained healthy, one was dead, and the others were severely injured. Eight of the nine plants inoculated with the isolate from lupine were diseased, and one remained healthy.

On June 23, 1949, 25 Dixigem peach fruits were used in an experiment to determine the ability of these isolates to attack peach. The hand-ripe fruits were washed with tap water, disinfected in $HgCl_2$ 1-1000 for two minutes, and then washed with sterile tap water. Five fruits were wounded with a sterile scalpel, and a small segment of sterile agar was inserted into each incision. These fruits were held as controls. In like manner, five peaches were inoculated with each of the three isolates used in the previous experiment and five with a recent isolate of the same fungus from a lupine seed. A small segment of agar from a vigorous culture of the isolate was substituted in each case for the sterile agar used in the controls. The point of inoculation was encircled with a moistened indelible pencil. The fruits were then held in moist chambers at room temperature. The controls were all healthy on June 27, whereas all inoculated fruit had typical anthracnose lesions two to three cm. in diameter at the point of inoculations. The fungus was reisolated from the lesions.

The results of the above experiments demonstrate that the isolates of G. cingulata from lupine and peach were capable of attacking both hosts. The extent of the damage that may result from growing a winter cover crop of lupine on or near a peach orchard is not known. The lupine, when used for green manure, is usually turned under in April or May, but when permitted to mature, seed is ready for harvest in late May or June. Anthracnose is most conspicuous on lupine stems and pods as they approach maturity although seedlings also may be killed. The presence of an abundance of spores in or near a peach orchard a short time before harvest would seem to be a potential danger to the fruit, either in the orchard, in transit, or on the market. Likewise, the presence of diseased fruit left in the orchard in July may constitute a menace to the lupine crop planted in October. Although at present no correlation between the amount of anthracnose in a peach orchard and the proximity of a lupine field has been shown, until more observations can be made it would seem wise to be cautious about planting lupines in peach orchards in sections where anthracnose is known to be present on either crop.

Literature cited

1. Dunegan, John C., and Joyce Kephart. Glomerella cingulata on peaches from Georgia and Maryland. U. S. Bur. Plant Industry Plant Dis. Reporter 33 (1): 18. 1949.
2. Ramsey, G. B., M. A. Smith and B. C. Heiberg. Anthracnose of peaches on the market. Phytopath. 38 (1): 22. 1948.
3. Weimer, J. L. Anthracnose of lupines. Phytopath. 33 (3): 249-252. 1943.

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND THE GEORGIA AGRICULTURAL EXPERIMENT STATION

JANUARY TEMPERATURES IN RELATION TO THE DISTRIBUTION AND SEVERITY OF DOWNY MILDEW OF TOBACCO II. A REVIEW OF THE PAST NINETEEN YEARS

Paul R. Miller and Muriel O'Brien

Blue mold's severe activity in 1949, following warm January temperatures, points out a similarity of behavior with several other years when blue mold was severe following warm January temperatures. In 1937[1] the first author published on the history of the disease and some observations on January temperatures in relation to the distribution and severity of downy mildew of tobacco. To bring the subject matter up-to-date, a review of the development of blue mold in relation to January temperatures for the nineteen years that the disease has occurred in the United States is attempted in this paper.

As is well-known, blue mold appeared in 1921 in the eastern tobacco area of Florida and Georgia. The disease was not then observed until 1931 when it appeared in the same region and spread northward as far as Maryland. The greatest spread and damage probably occurred in the years 1932 and 1933, while during 1934, 1935, and 1936 there was an apparent recession in both range and severity (Fig. 2). In 1937 the disease was again severe. Generally, the years 1938 to 1946 represented a period in which the disease was present but with no marked severity. In 1946 there seemed to be an increase in activity and severity of the disease with a high peak of severity reached in 1947. In 1948 blue mold was prevalent but characterized by mild spread with no great severity in any particular area. Blue mold in 1949 was very severe in North Carolina, South Carolina, and Virginia but was only moderately severe in Georgia and Florida. It is apparent, then, that blue mold activity has fluc-

[1] Miller, Paul R. January temperatures in relation to the distribution and severity of Downy Mildew of Tobacco. Plant Disease Reporter: 21: no. 14: 260-266. August 1, 1937.

References by States (Florida, Louisiana, Georgia, South Carolina) to the History and Severity of Downy Mildew of Tobacco, Peronospora tabacina Adam, for the past nineteen years, 1931-1949, as published in the Plant Disease Reporter.

Year and Volume	Florida	Louisiana	Georgia	South Carolina
1931:15 pp.	44-5	32,45	32,45,58	
1932:16	50	26	16,26*,27,50-1, 69*	52-3,55,69*
1933:17	28+,29		16,19,28+,29	16,19,28+,29-30
1934:18			6,49*	33,49*
1935:19	297*		36,104*,193*, 297*	104,193*,297*
1936:20	172		92,142	
1937:21 (1)(2)(3)	42+,43+,44+, 131,226-7,345	42+,43+	42,44+,107,132	43+,44+,108, 132-3
1938:22	60,202*, 204-5		60,70,88,101, 115,293-4	102,116,117
1939:23	83,383		51,112-3,177	112
1940:24				
1941:25			98-9,203	203
1942:26	223		121	
1943:27			174	208
1944:28	937		239-40,326	398-9
1945:29			201	270-1,390
1946:30			123,227-8	200
1947:31 (4)	122,198		121-2,150,199, 236,275-6	150-1,200,237
1948:32	132,168,236+		93,131,167,236+	168,233-4,236+
1949:33 (5)	239+	239+	166-7,239+	239+

For references and explanation of symbols see page 422.

References by States (North Carolina, Virginia, Kentucky, Tennessee) to the History and Severity of Downy Mildew of Tobacco, Peronospora tabacina Adam, for the past nineteen years, 1931-1949, as published in the Plant Disease Reporter.

Year and: volume	North Carolina	Virginia	Kentucky	Tennessee
1931:15:	43,44,57-8	61		
1932:16:	25,53-4,69*,70, 94*,96	94*,95		
1933:17:	19,28+,30-1,37, 60,163	28+,31,45, 60*		36-7,60*,104
1934:18:	49*	49		49*
1935:19:	104*,193*,297*	99,193*		104,193*,297*
1936:20:	136		188-9	56
1937:21: (1)(2) (3)	43+,44+,108, 133,184-5	27,43+,44+, 184-5,245+	44+,174,182-4, 227-8,246+	43+,44+,174, 245-6+
1938:22:	102,116,117,127, 182,202*,203	45-7,127, 203,252	163,184,382	128,182-4
1939:23:	88,112	139,153	176,191	
1940:24:	218-9		217-8,237	218
1941:25:		318		
1942:26:	111	52,432,434		
1943:27:	493	227-8,298-9,388 (correction),643		
1944:28:	398-9	482,848	451	423,451,483
1945:29:			209,719,440-1	
1946:30:		275-6,382	201,228-30,465	
1947:31: (4)	151,200-1,237-8, 324	238	239,427-8	239,276-7
1948:32:	168,234,236+, 305+	16-7,234, 236+,305+	234,236+,505	236+,348, 508
1949:33: (5)	239+	75,239+	239+	239+

For references and explanation of symbols see page 422.

References by States (Maryland, Pennsylvania, Connecticut, Massachusetts) to the History and Severity of Downy Mildew of Tobacco, Peronospora tabacina Adam, for the past nineteen Years, 1931-1949, as published in the Plant Disease Reporter.

Year and volume	Maryland	Pennsylvania	Connecticut	Massachusetts
1931:15	57,72,85-6			
1932:16	94	103		
1933:17	28+,60*	28+,60*,63,104		
1934:18	49*			
1935:19	193*,297*	193*		
1936:20	172			
1937:21 (1)(2)(3)	43+,44+ 331-2	43+	218	235,286-7,384
1938:22	128,182		182	181,204,251-2,327
1939:23	153			176,233-4,381-2
1940:24		216-7	216	216,257,299-301
1941:25				14,516
1942:26	243,477		201-2	7,286,337
1943:27				145,273-4
1944:28	714,753,838		714,903	
1945:29		558		
1946:30	202			
1947:31 (4)	238-9,277	277	22,278	278-9
1948:32	234,236+	305+,348	305+	305+,383,386
1949:33 (5)	239+	239+	239+	239+

For references and explanation see page 422

References by States (Ohio, Wisconsin, Indiana) and for Canada to the History and Severity of Downy Mildew of Tobacco, Peronospora tabacina Adam, for the past nineteen years, 1931-1949, as published in the Plant Disease Reporter.

Year and volume	Ohio	Wisconsin	Indiana	Canada
1931:15 to 1936:20 - no citation				
1937:21 (1)(2)(3)			235,246+	
1938:22				289-90
1939:23	177			
1940:24 to 1944:28 - no citation				
1945:29	558			
1946:30				
1947:31 (4)		419-20		239-40,279-80
1948:32				305+
1949:33 (5) ≠	239+	239+	239+	239+

Legend:
* - Summaries, notes; grouped mention of progress or severity.
+ - Dates and comparisons of first appearances.
≠ - Record through September 15, 1949.

Index to Special Articles:
(1) 1937:21: p. 47 - "Report of the Work of the Tobacco Disease Survey Committee for 1936"
(2) 1937:21 p. 130 - "A survey of the Tobacco Downy Mildew Situation in Florida, Georgia, South Carolina, and North Carolina, March 23 to April 20"
(3) 1937:21: p. 260 - "January temperatures in relation to the distribution and severity of Downy Mildew of Tobacco"
(4) 1947:31: p. 480 - "Can Tobacco Plant Beds in Kentucky and Tennessee be infected by Peronospora tabacina blown in from Texas?"
(5) 1949:33: p. 238 - "Tobacco Blue Mold in 1949 provides Lesson in Control: Status of the Warning Service to April 26th"

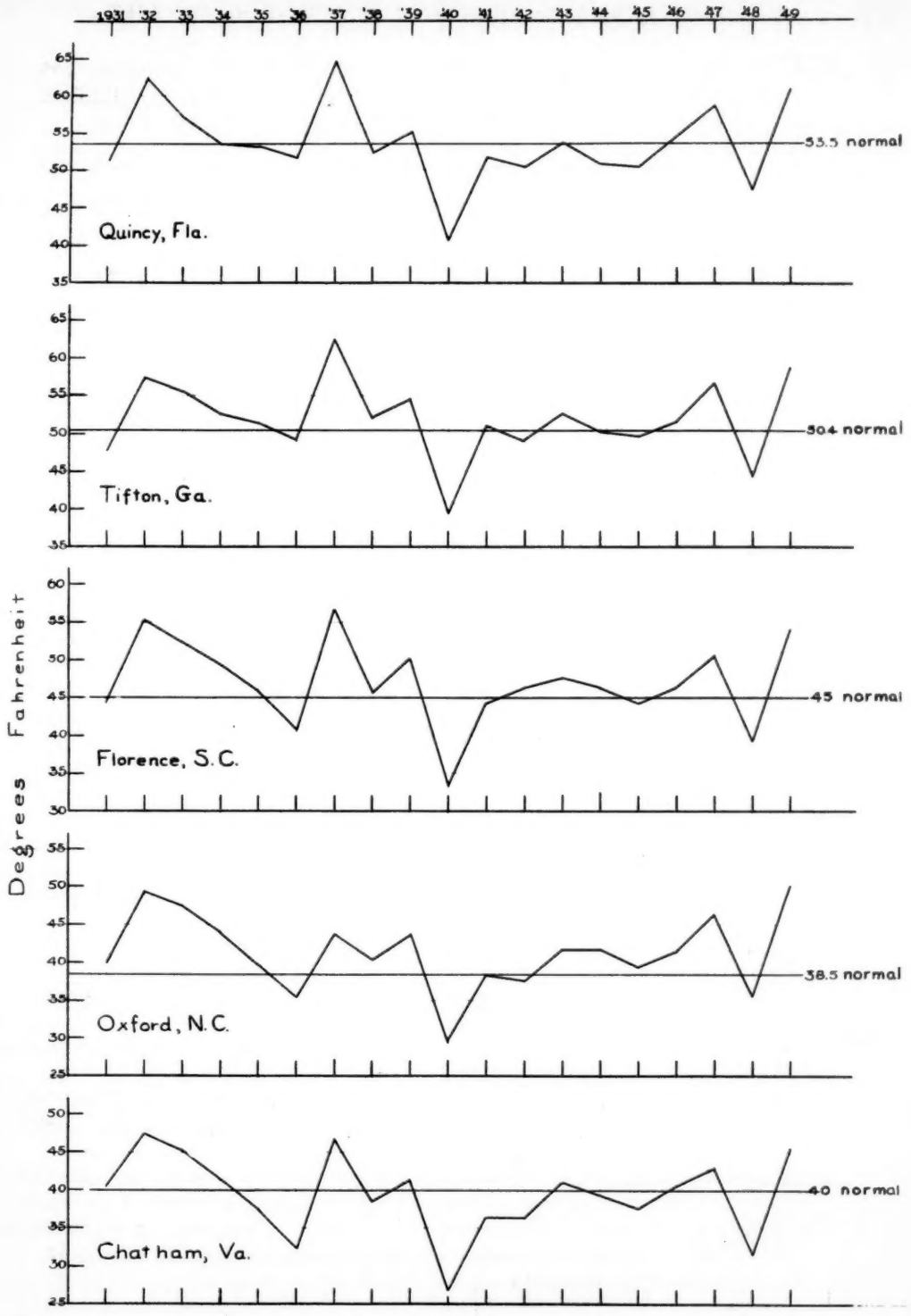

Fig. 1 - Mean temperatures for the month of January, 1931 to 1949 inclusive, at five Southern weather stations in tobacco-growing areas.

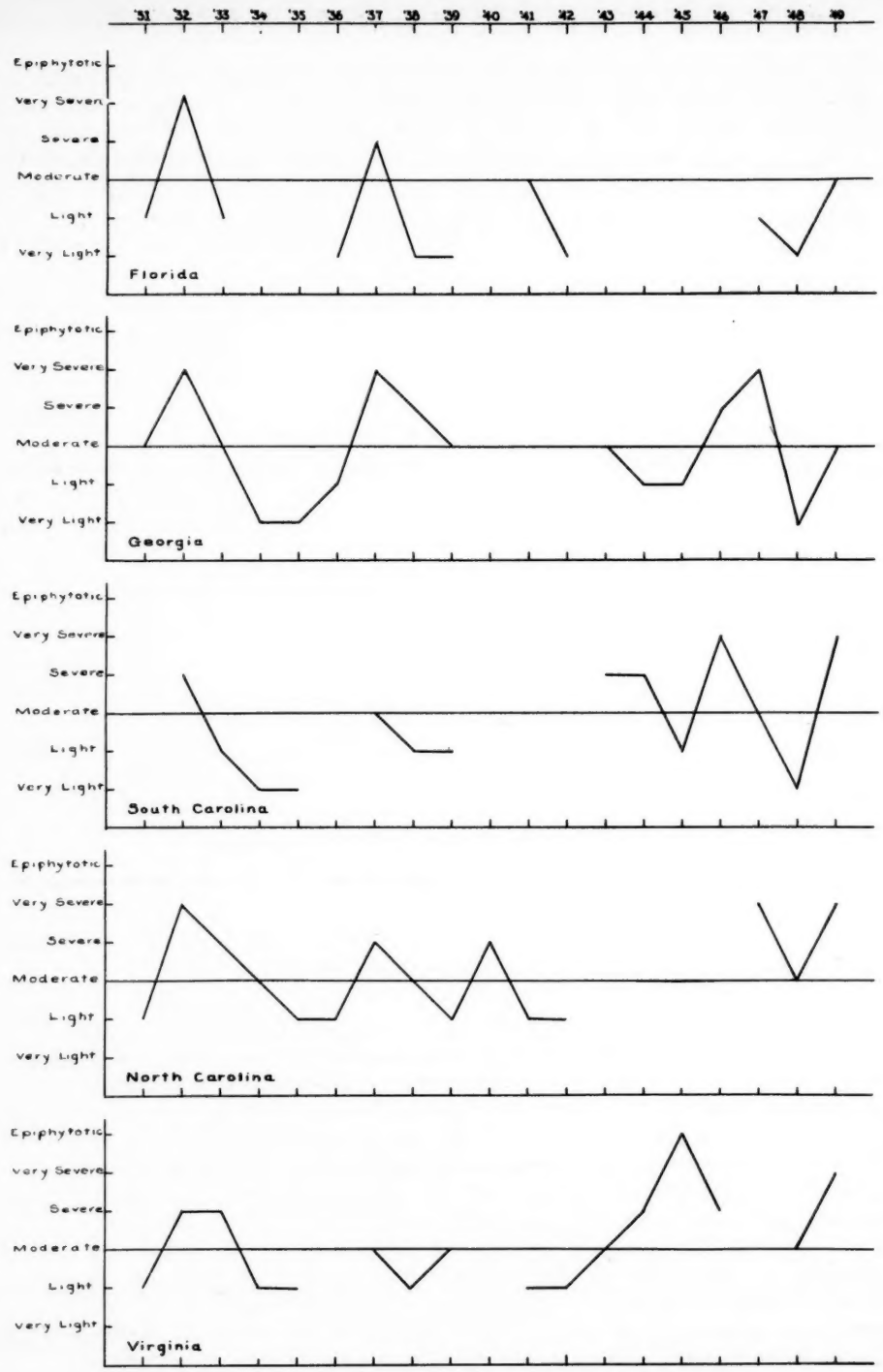

Fig. 2 - An Interpretation of the Severity of Blue Mold of Tobacco for the years - 1931 - 1949.

tuated greatly in both range and severity over the past nineteen years. There seem to be five peak periods when the disease was more active and severe, namely, the years 1932, 1933, 1937, 1947, and 1949. The reason for this fluctuation in intensity appears to depend upon late winter conditions and, in particular, January temperatures. In the years when mildew was most severe, January temperatures were above normal, whereas in the years when there was little disease activity, January temperatures were near normal or below.

Beginning with the first peak year, 1932, blue mold was quite active. January temperatures were, for the five locations shown in Figure 1, from +6.4 to +10.7 degrees above normal. Georgia for 1932 reported that there were no freezes or killing frosts in southern Georgia during the winter. The warm weather had afforded good conditions also for early plant beds and for growth. In 1933 the above-normal temperature range for the same places was +3.5 to a +9.1. Reports from various States during this year seemed to stress that while blue mold was severe, it seemed less so than in 1932.

Continuing the analysis of the peak years first, in 1937, after a lull in activity in the years 1934, 1935, and 1936, there was a renewal of blue mold activity. January temperatures were +6.6 to +10.7 degrees above normal. Blue mold was more widespread and in some cases more destructive. In Georgia the disease appeared very early, was severe in second-year beds, and was universally destructive. The early seasonal appearance of mildew was associated with the unusually mild winter, similar in some respects to that of 1932 when the disease appeared very early and caused heavy losses in many beds. It appeared again in western Virginia and western North Carolina, was widespread throughout Tennessee and the tobacco area of Kentucky. It was reported for the first time from Indiana, Connecticut, and Massachusetts. Along with Georgia, in the Carolinas and part of Virginia the disease was both prevalent and severe and caused heavy loss of plants.

In 1947 there occurred another January peak of above-normal temperatures, perhaps not quite as high as in 1937. In 1947, with a January temperature range of from +3.7 to +5.3 degrees above normal for the States shown in Figure 1, blue mold was observed in Georgia and Florida late in January. It was reported in the Carolinas, Maryland, Tennessee, Kentucky, Pennsylvania, Connecticut, and Massachusetts, and for the first time in Canada and in field tobacco in Wisconsin. Georgia reported a destructive year with an April estimate of over 70% loss.

The current year's peak resulted from January temperatures of from +6.0 to +9.2 above normal. Mold appeared in a Cook County, Georgia, new seedbed on January 11, the earliest seasonal observation ever made of the disease in a commercial Georgia seedbed. Mold was observed in the Florida shade tobacco area on February 11, two or three weeks earlier

than usual. In this shade tobacco area damage was more severe this year than in several years heretofore. In South Carolina blue mold occurred following "a considerable amount of warm weather in January and February!" A severe outbreak occurred in North Carolina with the disease fairly well-distributed in the old belt as far west as Guilford and Rockingham Counties. Virginia also experienced a severe outbreak.

In the years of light attack, going back to 1934, 1935, and 1936 in Figure 1, the mean temperatures were at the highest +5.5 and fell off to -7.5 from the normal for this three year period. There was likewise a recession in both range and severity of the disease for this period. In the years from 1938 to 1946 blue mold was present and active in varying degrees. In 1938 it was less severe than in 1937. Mean temperatures for January 1938 hovered about normal for all the southern points. Mold in this year was found in Georgia, Florida, in the Carolinas, Virginia, Maryland, Tennessee, Kentucky, and Massachusetts. North Carolina, South Carolina, and Georgia reported "no general outbreak"; Massachusetts that "it appeared in a few scattered beds but did not prove destructive". In Virginia it was widespread in the fields with slight damage resulting. On the whole blue mold did not develop so quickly following primary infection in all bed sites as it did in 1937. What damage did occur was probably offset by adequate recovery of plants before date of transplanting, by the advanced development of tobacco plants at time of infection, and by weather conditions generally unfavorable for the development of the parasite. It can be said that it was a little more severe than average but caused less damage than in 1937.

Temperatures in the years 1939 through 1946 hovered around normal with extreme lows in 1940. In 1939 the records show that in Georgia the epidemic was of moderate severity compared with '37 as a severe year and '38 as a light year. In Virginia it was considered more severe than in '38 but not as severe as in '37. In 1940 only North Carolina reported that it was "more serious than it has been since 1932". It was present in Pennsylvania in 1940 also where loss was about 25%, but failed to become destructive in Massachusetts, Connecticut, Kentucky, and Tennessee.

Nineteen forty-one and 1942 were negligible years with '43 showing an increase in activity of blue mold and loss of plants owing to freezes and the fungus. In 1944 Virginia experienced a brief epiphytotic; blue mold was severe in Williamsburg, Florence, and Horry Counties in South Carolina; light in Georgia, Connecticut, southern Maryland, and Florida. In 1945, a year with low January temperatures, there was some damage in Virginia and Kentucky. Temperatures in 1946 were normal to above. In Georgia and South Carolina blue mold was most destructive in attack and widespread in Kentucky and Virginia. Weather reports for the year indicate that conditions were ideal for blue mold development - warm January temperatures followed by an early spring. In 1948 blue mold was prevalent along the Atlantic Coast seaboard. It was mild in spread

with no great severity in any particular area. January temperatures were below normal for this year.

SUMMARY

A summary of all collaborator reports indicates that there have been five years out of nineteen that have been considered severe blue mold years. These are 1932, 1933, 1937, 1947, and 1949.

The yearly fluctuation in intensity of the disease has closely followed the line showing deviation from the normal of mean January temperatures at five southern points.

There seems to be a high coincidence between the intensity of blue mold in the five severe years and the above-normal January temperatures in those years. We do not know all the factors involved in this seeming relationship, but it is assumed that the above-normal January temperatures must stimulate oospore germination and consequently increase the disease potential. High temperatures of January apparently then determine the earliness of attack and build-up of inoculum. The maximum severity of the disease depends upon continuing favorable conditions for blue mold development as well as plant growth.

In presenting this review it is not intended that it be interpreted as the basis of a forecasting system. It is merely to point out some interesting relationships between January weather and the incidence of blue mold for the past nineteen years. It also suggests the need for epidemiological research which may explain the relationship.

DIVISION OF MYCOLOGY AND DISEASE SURVEY, CROP PLANT DISEASE FORECASTING PROJECT, BELTSVILLE, MARYLAND (Report of a study made under the Research and Marketing Act of 1946)

CUCURBIT DISEASES IN THE BLACKVILLE AREA OF SOUTH CAROLINA DURING 1949

Robert Aycock and Morris Hughes

Downy Mildew

The worst epidemic of cucurbit downy mildew (Peronoplasmopara cubensis Berk. & Curt.) [Pseudoperonospora cubensis (Berk. & Curt.) Rostow.] since 1938 (1) occurred this season. Symptoms were first noted on cucumbers May 30, about a week to ten days earlier than usual (1,2,3) and some 19 days after the first report of the disease in the State.[1]

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[1] Plant Disease Survey Warning Service Report No. 19, May 13, 1949.

Weather conditions were highly favorable for primary infection and secondary spread, so that the cucumber crop was completely killed in two to three weeks.

In spite of the destructive level which the disease reached on cucumbers, mildew caused little financial loss to most growers because of the collapse of the market in early June.

On cantaloupes, however, downy mildew became serious about a week prior to the first scheduled picking. The disease developed so rapidly that the foliage in many fields was completely destroyed in a few days' time. Since it is not a standard practice to apply protective fungicides to cantaloupes in this area, the crop was classed as almost a total loss. Farmers by their own estimates sustained losses in financial returns which ranged from 60 to 90 percent. These figures were based on the reduced yield of marketable fruit and the low price received for cantaloupes sold in spite of their extremely low quality.

In contrast to these figures yield increases up to 50 percent in the number of crates per acre were obtained in experimental dust plots at the Edisto Experiment Station. Moreover, these increases were obtained in spite of 5.5 inches of rain which fell on 15 days during the dusting period.

Downy mildew was also a limiting factor in watermelon production, although symptoms were not evident until early July. The delayed development of the disease in this area permitted a majority of the melons to reach the market before the disease became serious, but in the northern part of the State considerable defoliation occurred before the greater part of the crop was harvested.

Angular Leaf Spot

Angular leaf spot of cucumbers (Bacterium lachrymans E. F. Smith & Bryan) was not a major problem. Low rainfall and high temperatures during the early part of the growing season were responsible for keeping the disease at a low level. Two fields in an area which received additional local showers were noted as exceptions. Here an estimated 15 percent loss occurred due to leaf spot infection.

Powdery mildew

Powdery mildew (Erysiphe cichoracearum DC.), which usually appears too late to menace the cantaloupe crop (4), was first observed on May 25 and later, in June, destroyed most of the remaining leaves that survived the disastrous downy mildew epidemic.

Cantaloupe Mosaic

Mosaic caused by an unidentified virus was widely present in this locality. The most commonly observed symptoms were vein-banding and mottling of the younger leaves. Under field conditions the diseased plants appeared to be only mildly affected, although the severity of downy mildew prevented any accurate appraisal of the real effect of the virus on the host. No recognizable symptoms were observed on the fruit.

In one field near Blackville less than 3 percent of the plants showed symptoms in early May, but just prior to harvest 40 percent of them had become infected.

The disease is easily transmitted by the leaf-rubbing technique, symptoms appearing about six days after the inoculation of expressed juice.

Anthracnose

In a few localities anthracnose (Colletotrichum lagenarium (Pers.) Ell. & Halst.) was serious on watermelons. In Dorchester County, one field observed on June 8 had already became a total loss. Foliage infection had occurred at a high level and from 5 to 100 lesions were present on practically every fruit.

In the main watermelon areas, however, anthracnose occurred at a much lower level. The judicious use of Dithane and copper dusts by a steadily increasing number of growers is credited with markedly reducing the extent of infection.

Anthracnose also occurred to a limited degree on cantaloupes, but owing to the partial resistance of the Hale's Best Strains (4), which are widely grown here, little economic loss resulted.

Watermelon Wilt

Wilt (Fusarium bulbigenm var. niveum) [F. oxysporum f. niveum] continues to be one of the most important watermelon diseases. Heavy losses were sustained in some cases, even where the land had not been planted to watermelons for many years. One grower in Colleton County lost an estimated 30 percent of his stand by the last of May, in a field which supposedly had not grown melons for 14 years. Other fields which had not grown this crop for periods of from 5 to 8 years were observed in which as high as 50 percent of the plants were dead by harvest time.

High levels of infection such as these were observed in only a few instances but wilt was generally present to some extent over the whole area. The non-acceptance of available resistant varieties by growers indicates that this disease will continue to be a major problem in watermelon production for some time.

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EDISTO EXPERIMENT STATION, BRANCH OF CLEMSON COLLEGE, BLACKVILLE, SOUTH CAROLINA

MELON VIRUSES IN ARIZONA - 1948-1949

Paul D. Keener

During the past two seasons, limited surveys were conducted in the Salt River Valley in Arizona to ascertain the distribution and prevalence of various melon viruses. Included in the surveys were plantings of cantaloupes, representing the varieties Imperial 45, 45C and the newer sulphur-resistant forms, V-1 and SR-91. Areas of honey dew and watermelons were also examined.

With the exception of late-planted fields, mosaics were less widespread and severe in all melons in 1949 than in 1948. Although no general losses from mosaics were incurred by melon growers in the Salt River Valley during either season, several isolated centers of severe infection were noted in plantings of Imperial 45 cantaloupes and in honey dew melons. In 1949, a 40-acre planting of Imperial 45 cantaloupes in the vicinity of Glendale exhibited centers of infection running as high as 86 percent of the plants in any particular area, although the overall incidence of disease in this field did not exceed 37.5 percent of the stand. In a nearby 40-acre field, the percentage of mosaic-infected plants averaged 29.6. In both instances, the occurrence of symptoms of mosaics appeared to be coincident with the proximity of fende rows densely overgrown with noxious herbaceous and woody plants, as well as with fig trees exhibiting symptoms of fig mosaic. The experimental production in a greenhouse at Tucson of leaf abnormalities in a small number of summer squash seedlings after numerous cotyledon inoculations (carborundum-swabbed undiluted plant-sap method) with inoculum consisting of macerated mosaic-infected fig leaves, may have some significance. The apparent transfer of virus symptoms from a woody plant to squash may be a case similar to that reported by Moore, Boyle and Keitt (1).

In a late-planted 80-acre field 9 miles northwest of Phoenix, symptoms of a brilliant yellow-mottle type mosaic (squash mosaic ?) were observed in vines of Imperial 45 cantaloupe. This virus is apparently responsible for considerable distortion of leaves, flowers and fruits. From the same area, during the previous season (1948) symptoms of an apparently similar leaf-, flower- and fruit-distorting virus were observed in the same variety of cantaloupe. Macerated infected leaf tissues were used as sources of inoculum for squash, honey dew, and cantaloupe seedlings. Typical mosaic-like symptoms appeared in all inoculated seedlings in from nine to eleven days. These results are all the more significant in view of the cultural practice pursued by many small-scale growers in the Salt River Valley, namely, of growing squash adjacent to, or near, plantings of cantaloupes and honey dews.

The assumption that mosaics tend to build up in regions where melons are planted year after year is not substantiated by data collected for the past two years and from general observations extending over a period

of several years, in the Salt River Valley. In all melon plantings surveyed in 1949 just previous to harvest, symptoms of mosaics were noted in a trace to an average of 37.5 percent of Imperial 45 cantaloupe plants and in a trace to an average of 8.3 percent of honey dews. Corresponding percentages for 1948 were, 0.4 to 99 (cantaloupes) and 1 to 60 (honey dews). In an area where 99 percent of Imperial 45 cantaloupe plants became infected during the 1948 season, 29.6 percent of similar plants were affected in 1949. In isolated plantings in Southern Arizona in 1949, symptoms of mosaics were most noticeable in plants of honey dews, less evident in Imperial 45 cantaloupe, and were practically absent in plants of V-1 (sulphur-resistant).

Species of aphids occurring in the Salt River Valley are not completely known taxonomically. The reported paucity of all aphids in the region, including those known to vector melon mosaics elsewhere, may account for the comparatively low incidence of melon mosaics during the past season.

More pronounced and perhaps more significant than mosaics this year, in plantings of Imperial 45 and 450 cantaloupes and in honey dews, were symptoms usually attributed in most suscepts to curly-top virus. Whether or not this virus actually occurs to an appreciable extent in melons in the Salt River Valley has not been satisfactorily demonstrated. Both cantaloupes and honey dews are known to be susceptible to curly-top virus and field observations strongly suggest that melons may be generally affected in this State. Moderate amounts of curly top symptoms are observable every season in plants of watermelons in Arizona. Curly top has been on the increase in numerous hosts for the past several years.

Symptoms strongly suggestive of "yellow-net" virus (2) have been encountered in low percentages of cantaloupe and watermelon plants for the past two seasons in the Peoria and Buckeye regions. Further investigations are needed to establish the true nature of this disorder.

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CARROT BLIGHT CAUSED BY ALTERNARIA DAUCI APPEARS IN NEW MEXICO

Philip J. Leyendecker, Jr.

Bunching carrots[1] have been grown as a commercial irrigated crop in New Mexico well over 8 years. The discovery of Alternaria blight, caused by Alternaria dauci (Kuehn) Groves & Skolko, this year is the first major outbreak of the disease in New Mexico. One thousand to twelve hundred acres were damaged at Bluewater and Grants, the major carrot-producing area in the State. Approximately 200 acres were also damaged at Los Lunas, New Mexico, which is rapidly expanding its acreage devoted to carrot production.

In most fields where the carrots were ready for harvest, the older leaves were completely destroyed, leaving only two or three young leaves on mature roots. Packers estimated a loss of from 60 to 70 cents a crate because of increased harvesting costs and a 15 percent reduction in U. S. grade of the tops. The leaves in many fields were severely spotted and necrotic with large lesions on the petioles. Alternaria was consistently isolated from these diseased leaves and petioles.

All fields had been cropped to carrots previously, with the exception of one late 90-acre field which was planted to carrots for the first time and was isolated four or five miles from any other crop. Blight was very prevalent in this field, showing up on young as well as older leaves. Considerable damage to the roots as well as the tops can be expected. The prevalence of the pathogen in this isolated field could not be accounted for.

It is believed that the appearance of the disease in epiphytotic form was due to the unusually high amount of rainfall and the high humidity during late July and early August. Most areas in the State have received more moisture than usual during summer months of 1949.

The damage caused by the pathogen was checked temporarily by airplane dusting with a number of commercial copper fungicides.

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[1] Imperator predominantly, a few scattered acres of Morse's Bunching

ALTERNARIA LEAFSPOT OF GARDEN STOCK IN CALIFORNIA

Lily H. Davis, R. H. Sciaroni, and Frank Pritchard

In late June of 1946 a leaf-spot disease was observed[1] in commercial fields of garden stock, Mathiola incana (L.) R. Br., grown for cut flowers in San Mateo County, California. The disease was not particularly damaging at that time but growers reported severe losses later in that and the following year. It was again observed in October, 1948 and has since caused complete destruction of several small plantings.

The spots were round or elongate, 3-15 mm. across, pale grayish-green, concentrically zoned, especially in the older lesions that had brown centers; the spots became darkened by the superficial masses of nearly black spores. Lesions with water-soaked margins were also observed on the stems and flowers. The spots first appeared on the lower leaves and gradually spread upward.

Examination of the fungus from the host and in culture showed it to be Alternaria raphani Groves & Skolko (1). Neergaard (2) described the same fungus as Alternaria matthiolae, but in his attached sheet of "Additions" he states that the fungi are identical and that, since the name A. raphani antedates his, it should be used. The fungus is also identical with A. brassicae (Berk.) Sacc. var. macrospora Sacc. (2).

The first published report of the stock disease is that of Ware (3) from a greenhouse in England in 1935. Neergaard (2) mentions a mild attack of young stocks observed by Rostrup in Denmark in 1894; herbarium material showed this to have been caused by A. raphani. The fungus is widely distributed on Cruciferae, especially stock, radish, and cabbage in Denmark, Holland, Hungary, France, Germany, and Japan. It occurs on seed of these crops from many of these areas (2). In Canada and the United States (California, Michigan, Minnesota, New Jersey, Ohio, and Pennsylvania) (1) it has been found only on radish seed. Apparently this is the first report of the fungus on Mathiola in this country.

Alternaria raphani and the disease it causes have not been seen in commercial seed fields in Monterey and Santa Barbara Counties. Neither has it been seen in cut flower fields in the Los Angeles or San Diego areas. The higher humidity and cooler conditions of the San Mateo area may explain its presence there. The fact that the fungus is seed-borne probably accounts for the introduction of the stock pathogen to California. It is fortunate that the fungus does not occur in the California seed fields which produce much of the stock seed of the world. Indeed, it is questionable whether the fungus would ever become

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[1] Specimen, collected by K. F. Baker, in Herbarium, Division of Plant Pathology, University of California, Los Angeles.

established under the cultural conditions employed in seed production.

Cross-inoculation tests were made at the University of California, Berkeley, with Alternaria brassicae (Berk.) Sacc. sensu Bolle from cabbage and A. raphani from stock. The cabbage isolate was strongly pathogenic to cabbage but not to stock; the stock isolate was highly pathogenic to stock and caused minute dark lesions on cabbage. Neergaard (2) reports successful inoculations on Brassica spp., Cheiranthus cheiri, Iberis amara, Lactuca sativa, Raphanus sativus, and Mathiola incana, while Groves and Skolko (1) were successful in inoculating radish, cauliflower, broccoli, swedes, and cabbage. The latter was infected weakly, however, and inoculations on wallflower were negative. This inoculation evidence indicates that there may be physiologic races of the fungus. This is further corroborated by the fact that it has not been found in the stock seed fields in California but has been reported in radish seed from that State (1) grown in the same areas.

Good commercial control of this destructive disease has been obtained in cut-flower fields by applications of a dilute copper spray at 10 day intervals.

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DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, LOS ANGELES AND BERKELEY, AND SAN MATEO COUNTY AGRICULTURAL EXTENSION SERVICE, HALF MOON BAY

LEAF-BUD CUTTINGS AS A MEANS OF PROPAGATING DISEASE-RESISTANT ELMS

T. W. Bretz

The selection and breeding of elms resistant to phloem necrosis [virus] and the Dutch elm disease [Ceratostomella ulmi] have become increasingly important phases of the research on the control of these diseases. Of importance to the ultimate success of such work is the development of methods whereby resistant trees may be propagated readily -- particularly vegetative means that permit the production of resistant stock on its own roots. Relatively little work has been done on the vegetative propagation of Ulmus species through the use of cuttings and such data as are available are rather fragmentary and vague as to the exact procedures used. Investigations were therefore initiated a few years ago to develop reasonably simple, yet reliable, methods of increasing elms by means of cuttings. This is in the nature of a preliminary report on some of the methods used and the results obtained.

The initial attempts to root elm cuttings were patterned after the various methods described in the literature for the propagation of other woody plant species. With the exception of propagation by means of root cuttings, the results were generally unsatisfactory. The chief objections to propagation by root cuttings are the difficulties involved in getting the material and the limited number of cuttings that can be obtained from the parent tree without endangering its survival, if the tree is small. A potentially greater number of resistant individuals could be secured by using stem or leaf-bud cuttings from a resistant parent, and the use of such material has been stressed in these investigations. The various accepted methods for propagation by means of dormant stem cuttings gave inconsistent and generally poor results. The use of leafy stem cuttings in the usual types of propagation benches and chambers was also unsuccessful. Such material, even under conditions in which the relative humidity in the propagation chamber approached saturation, failed to hold up and after a few days wilted to the extent that it had to be discarded. It became evident that some means of maintaining the turgidity of the cuttings until roots could be initiated was a prerequisite to success.

Devices producing a fine mist-spray at normal, city water-line pressures, designed for use over open, vegetable display counters to keep green produce in a fresh, crisp condition, were installed over the rooting medium inside translucent, enclosed propagation chambers. Their height and spacing was such that complete moisture coverage of all cuttings in the chambers was insured. The misting heads were kept in constant operation after cuttings were placed within the enclosure; thus a supersaturated atmosphere around the plant material was maintained. Under these conditions, succulent, leafy cuttings remained completely turgid at all times, even under extremely high, summer, greenhouse temperatures, and

subsequent root development occurred in a high percentage of cases. Although the plant materials were kept moist, very little deterioration of tissues was experienced from rot-producing organisms. Maximum drainage of the rooting medium was provided to prevent waterlogging and to provide adequate aeration for the submerged plant parts. The rooting medium used was either river sand or puffed mica. Both gave satisfactory results.

There are many factors other than the environment of the cutting bench which are more or less important as factors influencing rooting of cuttings. The age of the tree, its environment, its apparent inherent potentiality for rooting, the source of the cutting material from an individual tree, the time of year at which cuttings are taken, and the use of "root-inducing" chemicals are a few which may be mentioned. In these investigations materials from many sources and of various types have been used. Collections have also been made at various seasons of the year and the cuttings have been given various treatments prior to insertion in the rooting medium. Greatest attention, however, has been given to material from known phloem necrosis-resistant Ulmus americana selections growing in nursery rows. The trees were approximately 8 years old.

Leaf-bud cuttings, consisting of leaf blade, axillary bud, and a shield of stem tissue, collected in May and June, have given the most uniformly satisfactory results. Cuttings were made from 17 different resistant selections in May. One series, which received no treatment prior to insertion in the rooting medium, showed an average of 60 percent rooting six weeks after planting. Another series of identical material, treated with a commercially prepared "hormone" dust before planting, showed an average of 83.52 percent rooting. Rooting was obtained in material from all selections when given the dust treatment, whereas two of the selections failed to root at all when left untreated. Collections made from 14 additional selections in June and receiving no treatment prior to planting, averaged 58.57 percent rooting after six weeks. Identical material given the dust treatment prior to planting averaged 88.57 percent rooting. Some rooting was obtained in all selections when treated, but one selection failed to root in the untreated series. The primary roots branched profusely within a few weeks. In some instances, growth of the axillary bud began immediately and a new individual was well developed by the time the cuttings were removed from the rooting medium. More frequently, however, the buds remained dormant and did not begin growth for some time after the cuttings were removed from the propagation bench.

These results suggest that with phloem necrosis-resistant American elm, classed as "difficult" to root by more conventional means, leaf-bud cuttings, treated with a "hormone" dust and held under constant mist, provide a satisfactory means of increasing such stock on its own

roots. Potentially, a new individual can be secured from each node of the current years' growth, with this technique, thereby affording a rapid means of multiplying particularly valuable or desirable trees. The method has been successful with miscellaneous collections of Ulmus americana, U. pumila, U. thomasi, and U. fulva. Successful rooting of the Buisman elm has not yet been secured. Limited trials with hybrid Castanea (chestnut) have been successful. Further survey work on the utility of this propagation technique for other deciduous and evergreen trees is planned.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, IN COOPERATION WITH THE MISSOURI AGRICULTURAL EXPERIMENT STATION, COLUMBIA, MISSOURI

THE PRESENT KNOWN DISTRIBUTION OF OAK WILT IN MISSOURI

T. W. Bretz

Oak wilt, caused by the fungus Chalara quercina, was first found in Missouri in 1944 in a St. Louis residential area.[1] In 1946 it was reported from Mexico, Missouri (Audrain County) and at a number of new locations in and around St. Louis.[2] The proximity of the disease to the heavily timbered Ozark area, where oaks constitute the dominant tree species, has been of much concern to informed persons interested in forest problems in this region. During the past summer oak wilt was found in a number of new locations in central and southern Missouri. The trees from which specimens were taken showed typical oak wilt symptoms, and the fungus was obtained from collections made in Warren, Callaway, Boone, Randolph, Miller, Camden, and Butler Counties. The present known distribution of the disease in Missouri is shown in figure 1.

In the Clark National Forest in Butler County, an area of about 2/5 of an acre was involved. All trees in the stand, which consisted of scarlet and black oaks, were dead or dying. Mr. J. M. Nichols, Superintendent of the University of Missouri Forestry School Summer Camp, first observed this affected area in 1948 and estimated that it had doubled in size during the 1949 season. In a wood lot composed chiefly of pin oak in Randolph County, the disease is also well established and probably will eliminate this species in the stand.

All of the 1949 collections, with the exception of that made in Butler County, were made adjacent to main highways. In the majority of locations only one or a few diseased trees were found, although oaks killed in previous years could usually be found in the immediate vicinity. Thus, the number of cases of oak wilt found this year may very well represent only a small percentage of the actual cases that exist in the areas included in this report. Further scouting probably would result in the discovery of additional oak wilt stations in the present known infested areas as well as in other sections of the State.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. D. A., IN COOPERATION WITH MISSOURI AGRICULTURAL EXPERIMENT STATION

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[1] Bretz, T. W. Finding of oak wilt in Missouri. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Reptr. 28: 951. 1944.

[2] Denning, J. A. New locations and new host records for oak wilt in Missouri. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Reptr. 30: 32. 1946.

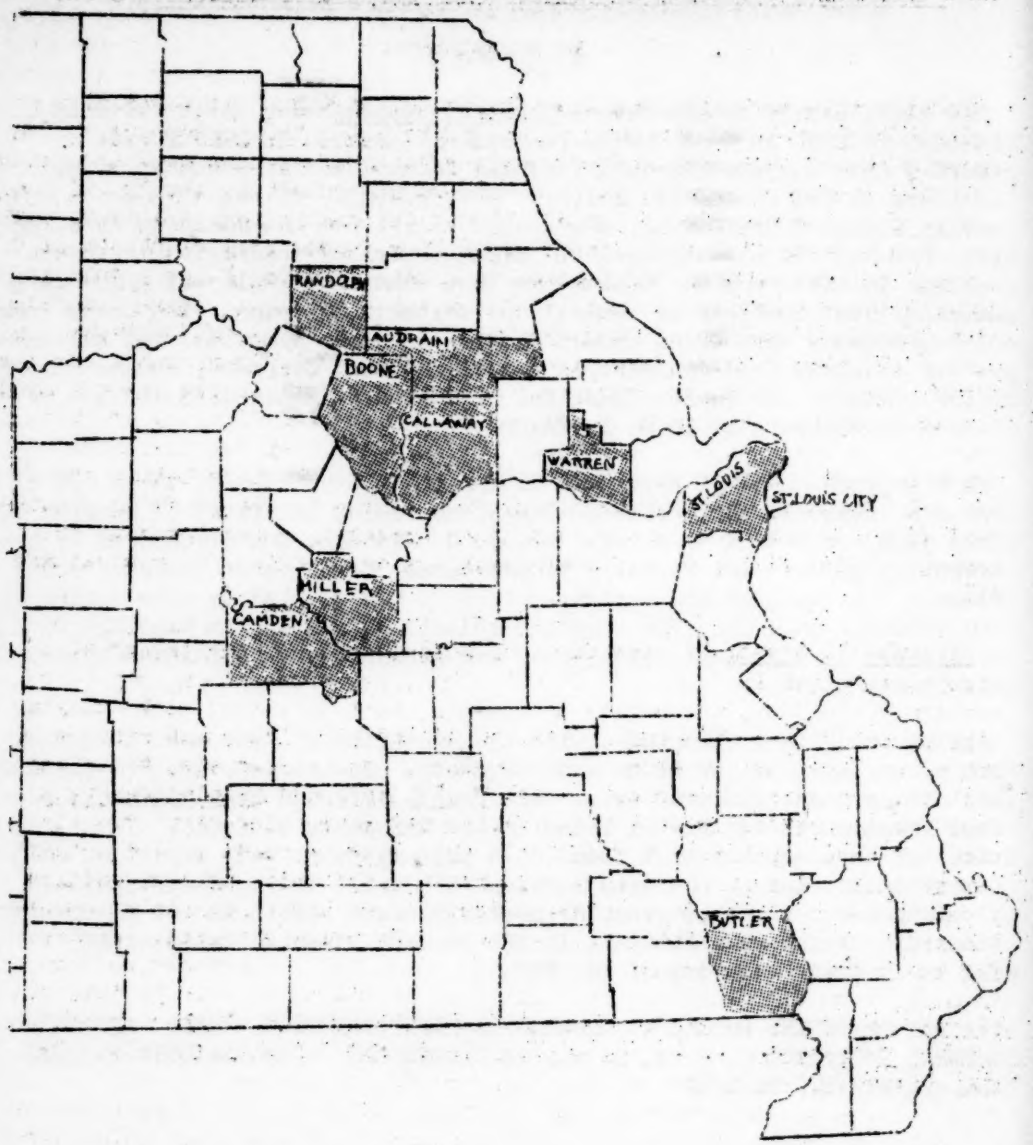


Figure 1. The Present Known Distribution of Oak Wilt in Missouri

PRESENCE OF THE TWO MOST DESTRUCTIVE RICE PATHOGENS IN COSTA RICA

Lucy Hastings

Considerable work has been done during the past year in Costa Rica on the seedling phase of the disease caused by Helminthosporium oryzae Van Breda de Haan. Germination and seedling pathogen studies have indicated that the spores of this fungus are carried on and within the interior of the seed to a rather heavy extent. Under the conditions of a number of greenhouse tests it was the important factor in reducing plant stands for several collected rice seed lots of the Fortuna, Nira, Rexere, and several criollo types commonly planted in the American tropics. These investigations included studies of seed samples from Costa Rica, El Salvador, and Guatemala. Fungicidal evaluations gave highly significant increases for Ceresan-treated seed for stand data and plant weight in the several seed treatment tests. No other treatments controlled the disease, the marked symptoms of which make it a very interesting and easy organism with which to work. Within five days after emergence symptoms became apparent. At first these are brownish discolorations on the seedling sheaths, oval in shape with reddish-brown margins, becoming ash-colored in the center. Spots coalesce, affected seedlings are girdled, turn brown and die.

Piricularia oryzae Br. and Cav. has been found on Magnolia and Nira rice plants sent in from plantings in the large rice-growing region of western Costa Rica, where this disease has been estimated to be causing considerable loss. The writer has looked for this important rice pathogen since the start of these investigations over a year ago, believing that the economic importance of both Piricularia and Helminthosporium could become rather great with the presently increasing plantings of rice in these equatorial regions. The Piricularia needs a high humidity for secondary infection (conidia are profusely formed under humidities close to 100 percent but only negligibly formed under much drier conditions). Here, in Turrialba, for example, the average relative humidity for the current rice season is above 89 percent. As observed on rice in Costa Rica, this disease is a blast or sterility with infections at the joints of the plants causing stalks to break with shattering of seeds. Infections are on spikelet pedicels and in the spikelets. In the spikelets, the lemma and palea are perfectly formed but other flower parts are entirely missing or dwarf and diseased. Isolations of Piricularia oryzae have been made from plants with all these parts diseased, and tests have been set up to study possible methods of control through seed treatment. To what extent this disease is seed-borne has not been clearly demonstrated. The fungus was easily isolated from seed of these diseased plants. Because the disease outbreak seems to occur very generally on the spikelets it would seem that the seed-borne aspect of the disease may be very important. Other, perhaps associated, fungi include Helminthosporium, Cladosporium, Curvularia, and Fusarium species.

Seedling phase symptoms appear less clearly defined than those of Helminthosporium. It would appear that a considerable toll can be taken by this fungus in the seedling stage, apart from the tremendous losses in yield due to Piricularia when it is prevalent.

The environmental conditions in many parts of Costa Rica are such that there are indications that both these diseases are potential problems or perhaps already problems, not entirely recognised since the extent of replanting and losses in plant stand and yield have not been studied. Further work on these two diseases and other rice pathogens is in progress which is to be reported more fully at a later date.

INTER-AMERICAN INSTITUTE OF AGRICULTURAL SCIENCES, TURRIALBA, COSTA RICA, October 5, 1949

BRIEF NOTES ON PLANT DISEASES

ROSEN'S BACTERIAL STALK ROT VS. ELLIOTT'S PYTHIUM STALK ROT OF CORN

By G. H. Boewe

The occurrence in unusual abundance over a wide territory of rot of the basal parts of corn stalks has been reported in midwestern States this year. (Ullstrup, PDR 33: 331. Aug. 15, 1949; Valleau and Diachun, PDR 33: 341. Sept. 15, 1949; Boewe, PDR 33: 342-343. Sept. 15, 1949). Ullstrup and Valleau and Diachun attributed the disease to Pythium butleri Subr.; Boewe, on the basis of field examinations and limited cultures, diagnosed it as Rosen's disease.

Field symptoms of the disease in all cases appear strikingly similar: the rotting of the lowermost internodes, followed by complete retting of the rind and pith of the stalks, falling of the stalks, and twisting of the vascular bundles in the rotted region as the stalk falls.

Complete agreement as to symptoms, the upsurgence of the disease in contiguous States under unusual and similar weather conditions, and its tendency to appear most abundantly in fields on low ground, all point to the probability that one and the same disease is the subject of all three reports.

As to the confusion between Elliott's Pythium rot and Rosen's bacterial rot, it is interesting to note that Valleau and Diachun readily obtained Pythium from their material while we obtained none in a limited series of cultures in which it should have appeared if present in the cultured material. Perhaps the two reported diseases need further, very careful comparative study.

ILLINOIS NATURAL HISTORY SURVEY, URBANA, ILLINOIS

SCLEROTIUM BOLL ROT OF COTTON

By James A. Lyle

An unusual type of damage to cotton bolls by Sclerotium rolfsii Sacc. was recently observed. This boll rot was noticed in a field in Escambia County, Alabama, in early August, 1949. The plants were luxuriant and the growth was dense. Only fully grown bolls were attacked. These bolls were located from 1.5 to 2.0 feet from the soil surface. Mycelial mats and sclerotia were formed on the pedicels, the split carpels, and the immature lint. In a few instances plants had been killed by infections near the soil line.

Sclerotium rolfsii occasionally causes damage to cotton plants, but it is not a serious threat to the crop in Alabama. It is unusual to find the fungus attacking cotton bolls, or any other fruit, more than a foot above the ground.

ALABAMA POLYTECHNIC INSTITUTE, AUBURN

NOTE ON OAK WILT IN GARY, INDIANA

By J. C. Carter and George Kuny

In view of the fact that oak wilt is spreading very rapidly and becoming extremely important, it seems desirable to add what is an apparently earlier record of the occurrence of oak wilt in Indiana to the report given by Dr. George B. Cummins of the Purdue University, Agricultural Experiment Station, Lafayette, Indiana, in the Plant Disease Reporter of August 15, 1949.

On June 28, 1949, Mr. George Kuny, Chief Forester of Gary Parks, Gary, Indiana, whom we have known through our contacts with Park Executives in Chicago and the Midwest Institute of Park Executives, sent in for laboratory examination samples of diseased oak trees. We cultured this material and obtained the oak wilt fungus Chalara quercina Henry from it and so reported to Mr. Kuny on July 21, 1949.

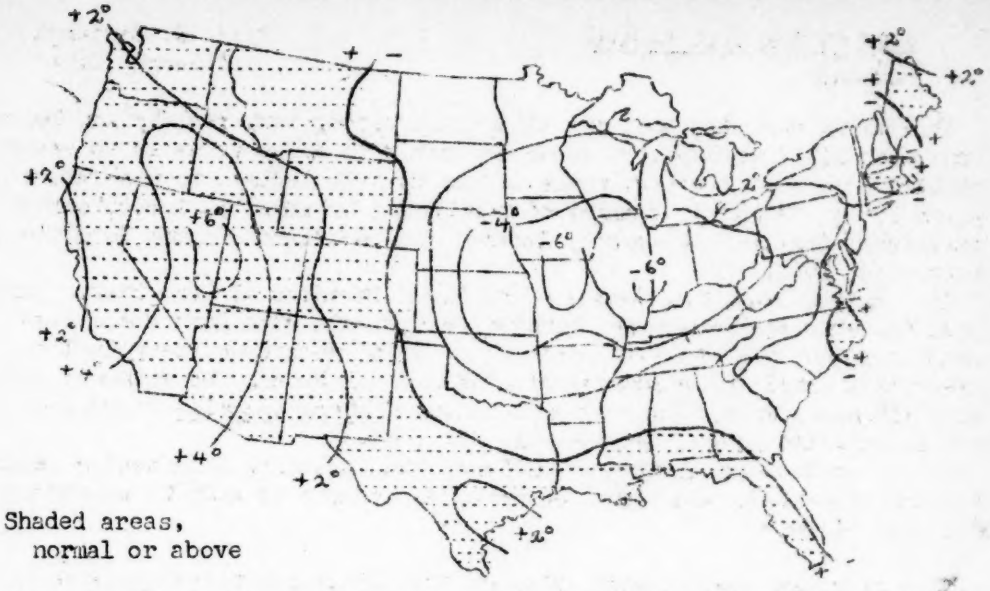
At our request Mr. Kuny has furnished the following information regarding the above trees and other occurrences in Gary of what is apparently the same disease:

"The branches sent in were obtained from black oak trees, growing in a private yard in the Glen Park area of Gary. The disease was first noticed a year ago on one of the oaks in a group of apparently healthy, virgin black oaks. At that time, the tree was treated by a commercial 'tree expert' for iron deficiency and was highly fertilized. The condition did not improve, but the tree was allowed to stand until this spring. When it failed to leaf out, it was cut down. Since that time, five black oaks, whose leaves almost touched the first diseased tree, have died. These were removed, stump and all, hauled away and burned. Others in the same area are now dying, starting at the top, and within two or three weeks, are completely dead."

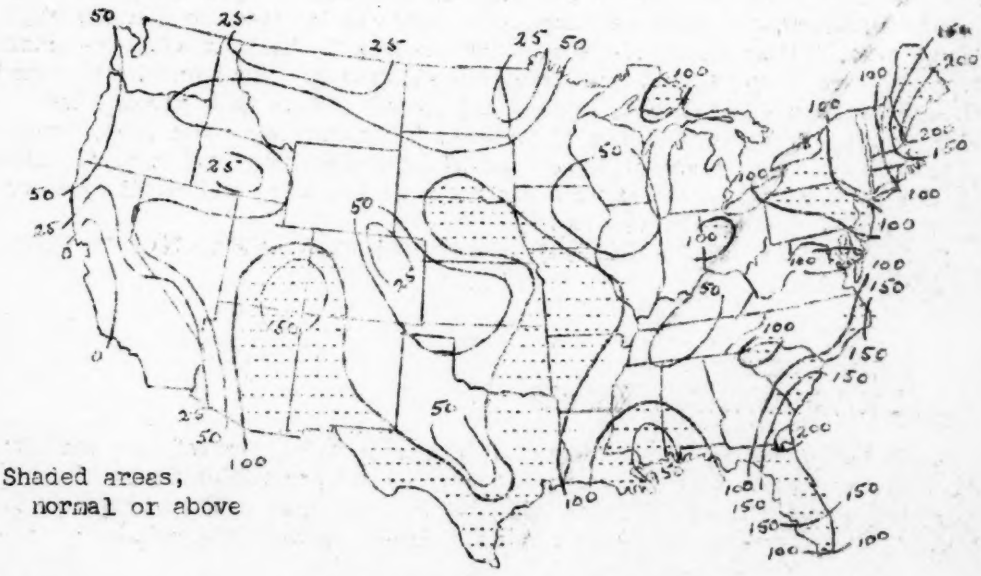
ILLINOIS STATE NATURAL HISTORY SURVEY, AND GARY PARKS, CITY OF GARY, INDIANA

A CORRECTION: WHEAT STREAK-MOSAIC VIRUSES

H. H. McKinney calls attention to an error in his article on varietal reaction to wheat streak-mosaic viruses in the preceding issue of the Reporter (vol. 33, no. 10, Oct. 15, 1949). On page 369, paragraph 3, line 3, (ros. immune sel.) should read (ros. susceptible sel.).



Map I. Departure of mean temperature from normal for September, 1949



Map II. Percentage of normal precipitation for September, 1949

SEPTEMBER WEATHER

(From U. S. Department of Commerce, Weather Bureau, Weekly Weather and Crop Bulletin for the week ending October 4, 1949)

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